Short report

Finger tremor in Parkinson's disease

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SUMMARY Finger tremor was investigated in 20 patients (age range 54-88 yr) diagnosed as suffering from idiopathic Parkinson's disease and six controls of a similar age and no known neurological abnormality. In nine of the patients tremor was not clinically obvious. When the tremor of these patients was recorded immediately after voluntary movement and subjected to instrumental analysis there were consistently observable differences from the controls. Such analysis may have diagnostic potential when there is clinical uncertainty. Surface EMG recordings were obtained from four patients. One patient had a large resting tremor with obvious reciprocating activity in flexors and extensors; in the others who had no symptomatic tremor there was reciprocating activity only after movement, and this died away in a few seconds as the induced tremor disappeared.

The diagnosis of Parkinson's disease is made when at least two of the principal features of tremor, rigidity, bradykinesia, and impaired righting reflexes are present. It is essentially a clinical judgement; there is no simple diagnostic test. The resting tremor of Parkinson's disease has been extensively studied and has characteristically a high amplitude and a very stable frequency of 4-6 Hz.¹ It is therefore very different from physiological tremor which is a low amplitude tremor usually with a peak at 8-12 Hz.² Recent work has demonstrated that the musculature in man and animals is thixotropic in nature and a suggested role of this time dependent stiffening has been an increase in postural stability and a consequent reduction in tremor.³ The main purpose of this study was to discover whether, in Parkinsonian patients without clinically obvious tremor, a tremor could none the less be revealed by manoeuvres designed to overcome this thixotropic stabilisation.

Methods

Twenty patients were investigated, of whom 14 were men. The age range was 54-88 yr (mean 73.5). All patients were instructed to omit their medication prior to the study.

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Tremor was also assessed in a control group of six patients, without any known neurological problem, who attended a day hospital. Their age range was 74-90 yr (mean 80.3). The patients' informed consent was obtained, and the study was approved by the local ethical committee.

The relaxed index finger was attached to a sensitive velocity transducer by means of a light crank. The forearm and hand were comfortably supported, and as movement occurred in the horizontal plane no sustained muscle activity was required. Recordings were always made immediately after a 10 s period of voluntary movement. The velocity was recorded at high gain over a 5 s period on disc and subsequently subjected to fast Fourier analysis to provide the frequency spectrum of the tremor. The sensitivity of the transducer was 8.1 V rad s⁻¹ and the peak-peak velocity of the tremor recorded after movement was usually in the range 0.1-1.0 rad s⁻¹. Results with this apparatus and normal young subjects have been described; there is normally no recordable rhythmic tremor at rest.⁴ Immediately following a short period of voluntary movement some activity is seen, examination shows it to result from the semi-regular discharge of a few individual motor units. This activity subsides in 20-30 seconds and the movement ceases. There is a possibility that there is a low level underlying rhythmical activity present in the Parkinsonian patients and that this is merely enhanced by movement. To check this in four patients measurements of surface EMG were made using suction cup electrodes (SLE Ltd) before and after movement.

Results

Patients with a large rhythmical tremor

Eleven patients had a large amplitude tremor. This

Fig (a) Upper trace. Tremor from a patient with a large rhythmical tremor. Frequency analysis (lower trace) reveals a large sharply tuned peak at 4.5 Hz and a harmonic at 9.0 Hz. (b) Upper trace. The enhanced tremor of a patient with no resting tremor. The tremor, which rapidly declines, is much smaller than in (a). Frequency analysis (lower trace) reveals a peak at 4.7 Hz and harmonics at 9.4 Hz and 19 Hz. The peaks, although smaller than in (a), are sharply tuned.

tremor was obvious to the patient and observer, and as expected it had an amplitude that varied although it was never entirely absent, and a very stable frequency. (fig. a) It is noteworthy that three of the patients were able to suppress the tremor by careful positioning of the limbs under normal circumstances but this ability was lost when they were coupled to the apparatus. As would be expected from a regular but non-sinusoidal oscillation, frequency analysis revealed the presence of harmonics (cf¹) and a sharply tuned fundamental which was at about 5 Hz. The fundamental frequencies are summarised in the table. Short periods of voluntary movement in this group of patients tended to produce a long lasting increase in the amplitude of the tremor but no change in the rate. As expected,

some of these patients found it difficult to initiate the required voluntary movements. In one of these patients EMG recordings were made. Rhythmical reciprocating activity was obvious; it was not changed significantly after a period of movement.

Patients without observable tremor

In nine patients tremor was not noted upon clinical examination, nor did the patients complain at any time of tremor. Analysis of records obtained from this group confirmed that there was no rhythmical tremor at rest. However, the situation was different following voluntary movements. The patients were asked to perform a series of flexion/extension movements of the finger before coming to rest in a comfortable position. Recordings of tremor were made for 5 s immediately after these voluntary movements. Rhythmic tremor was now recorded from all but one patient, and frequency analysis revealed a peak at about 5 Hz and, commonly, harmonics (fig. b). Predictably the amplitude of the tremor peaks was smaller in this group than in the patients with a readily visible tremor, but the characteristic frequency and the sharpness of tuning were similar. The fundamental frequencies and amplitudes of the peaks are summarised in the table. The one patient who failed to show this rhythmic tremor was a man aged 57 yr; his other symptoms were mild. Surface EMG recordings were made in three of these patients. There was normally no recordable rhythmic activity, but after movement some

Table Tremor frequency (Hz)

Patients with visible tremor	Patients without visible tremor
5.4	6.2
4.8	4.7
4.6	4.2
6.4	5.9
4.5	5.6
4.8	5.6
5.3	5.1
4.9	5.5
5.7	
5.0	
6.1	
Mean 5.2	5.4
SD 0.61	0.66

reciprocating activity was noted which died away as the tremor disappeared.

Age matched controls

The aperiodic series of movements that typically follow a brief series of voluntary movements of the finger have been previously described.⁴ Rhythmic tremor is rarely seen and where it occurs it is at a frequency of 10–12 Hz. These results had been obtained in relatively young subjects; as Parkinson's disease is primarily a disease of the elderly an age matched control group was studied. In this group of patients there were a few twitches after movement and Fourier analysis failed to reveal any rhythmic component. The results were very similar to the previously studied younger control group.

Discussion

A technique has been described which reveals a rhythmic 4–6 Hz tremor in a group of Parkinsonian patients in whom the tremor is not clinically obvious. The tremor does not occur at rest but can be observed immediately after voluntary movements. Tremor is sometimes classified as intention, resting and action tremor. This type of tremor does not precisely fit any of these descriptions; it might be described as activated or enhanced pathological tremor. Under these circumstances, tremor analysis may serve to strengthen an uncertain diagnosis of Parkinson's disease. It might also be of use in providing an early indication of the onset of the iatrogenic form of the disease. In control subjects there is also an increase in instability following movement, but in this case there is no rhythmic tremor. These observations suggest that in the group of patients without observable tremor the abnormal mechanisms for generating a pathological 4–6 Hz tremor exist, but do not normally come into play. In the other 11 patients there was a marked rhythmic tremor of large amplitude. In this group voluntary movement failed to change the frequency of the oscillation although it tended to increase the amplitude. Conversely, it is a common observation

that by firmly holding the affected limb immobile the tremor can often be felt to die away. Patients can sometimes exploit this to conceal a tremor. The experiments described here suggest that prior voluntary movements can provoke a tremor. There are several possible causes for this effect. One is that the effort of producing the movements causes the tremor; this is unlikely as similar movements of the contralateral hand did not do so. Secondly, movements may increase the excitability of the motoneuron pool for some time afterwards perhaps by altering Renshaw inhibition. A third possibility, which is being actively explored, is that the effect of movement is to cause postural instability because it decreases the stiffness of the musculature,^{3,5} and this allows the pathological tremor to exert itself until thixotropic restabilisation occurs. Prior movements of the limb can unmask a pathological tremor; this may be due to the elimination of muscle thixotropy.

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